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Bifurcations in the Impedance Cardiograms of Apparently Healthy Men and Women

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Key Words. Stroke volume · Cardiac output · Age · Body build · Posture

Abstract. We have compared data from apparently healthy subjects who produced an impedance cardiogram (ZCG) having a bifurcation in the dZ/dt wave with data from other subjects to determine whether data from the bifurcated waveform should be extrapolated to the general population. Thirty men and 30 women, 21–59 years, participated. The 8 men and 5 women with bifurcations in their ZCGs were shorter, with more ponderous builds and larger chests and abdominal circumferences, and they had lower respiratory flow rates than subjects with normal ZCGs. They also had several differences in cardiovascular function at rest and in response to postural change that were measured independently of the ZCG waveform. Furthermore, the men with bifurcated ZCGs had lesser changes in stroke volume as determined from the ZCG when they changed posture than the men with normal ZCGs. We cannot explain these waveform differences by the physics of impedance or by pathology. The possibility exists that these waveforms indicate something other than 'normal'. Thus, we recommend that cardiovascular data derived from impedance cardiograms with bifurcated dZ/dt waveforms not be extrapolated to the general population until we understand the etiology of the waveform.

Introduction

Impedance cardiography is a noninvasive technique for determining stroke volume. It is based theoretically on Ohm's law and the principles of volume conduction of electricity [1–4]. It has many advantages for applied physiologists who study cardiovascular control mechanisms in human subjects [3, 4]. Besides being noninvasive and safe, it allows for continuous, beat-by-beat monitoring of stroke volume and cardiac output. It demands no active participation by the subject. And it provides complementary cardiovascular data such as systolic time intervals and changes in thoracic fluid volume. The equipment is lightweight and convenient. Stroke volume and cardiac output data from this technique are repeatable and correlate well with data from other techniques such as electromagnetic flow probe and dye dilution [3, 4]. Relative changes in stroke volume and cardiac output

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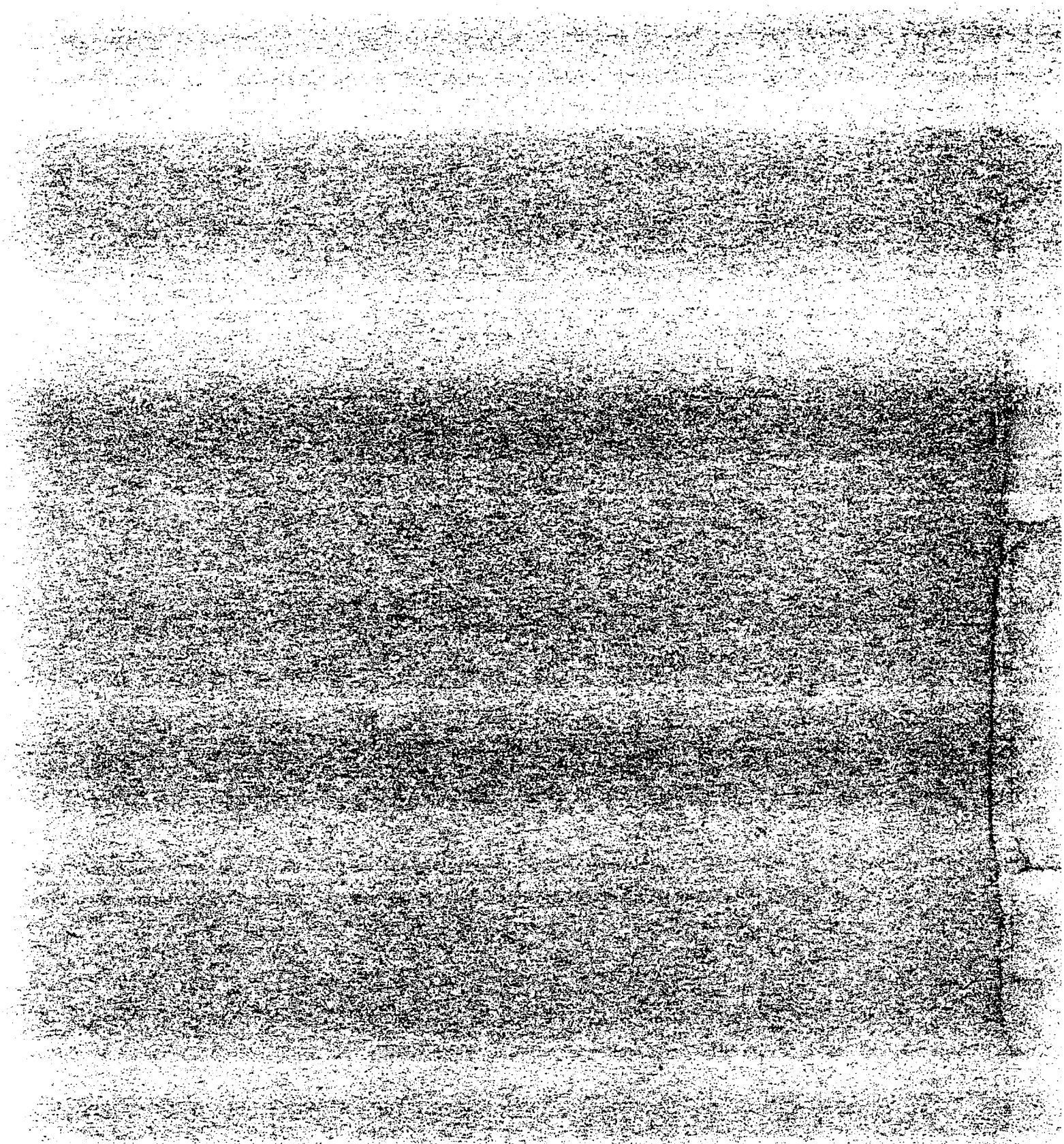


within an individual as measured by impedance cardiography correlate even better with other techniques than do absolute values [3, 4].

However, impedance cardiography has shortcomings. As with any indirect technique, it is based on assumptions. The physiological events that produce typical waveforms are reasonably well identified; however, aberrations in the waveforms of certain cardiovascular patients render the technique unreliable for determining stroke volume [3].

We have occasionally observed aberrant impedance cardiograms from subjects who appear to be in good health as determined by history, physical examination, laboratory tests, and treadmill stress test. The most common anomaly is a bifurcation (double peak) in the portion of the impedance cardiogram that corresponds to cardiac ejection. The necessity for reliable data and the importance of this technique in applied physiology dictate that we understand the functional significance of this waveform variation and the validity of impedance cardiography data from these subjects.

The purposes of this study, therefore, were (1) to explore the etiology of these aberrant waveforms from apparently healthy men and women by evaluating physical, anthropometric, respiratory, and cardiovascular data from subjects producing them and comparing these data with data from subjects producing typical impedance cardiograms, and (2) to determine whether data derived from such waveforms should be generalized to a normal healthy population.

Methods

Overview

Thirty men and 30 women were monitored with impedance cardiography and other cardiovascular monitoring techniques while in supine, seated, and standing postures. Each subject's impedance cardiograms were classified as normal (type I) or aberrant (type II). Physical, anthropometric, respiratory, and cardiovascular characteristics, and responses to change in posture, of subjects producing type I waveforms were compared with those variables of subjects producing type II waveforms. The men's data were analyzed separately from the women's data.

Subjects

Approximately equal numbers of men and of women in each of the four decades from 20 to 59 years participated. We recruited men and women of varied anthropometric characteristics who were nonsmokers and were taking no prescription medication except oral contraceptives or estrogen therapy by some of the women. Two of the men had participated in a previous investigation and had been identified as having type II waveforms; all other subjects were recruited without knowledge of their impedance waveform type. The men were tested approximately 6 months before the women. The subjects completed a medical history form, which was reviewed by a physician, and, if the subject had no medical or surgical problems that would preclude safe participation, he or she was cleared for the study. The study was approved by the Kennedy Space Center Human Use Review Committee, and subjects signed a consent form after being fully informed about all procedures, techniques, and risks.

Procedure

Subjects reported to the laboratory 1–2 h postprandial, and we recorded the following measurements: height; weight; chest circumference; abdominal circumference; and skinfold thickness of the biceps, triceps, subscapular, and suprailiac areas. Percent body fat was determined from skinfold measurements using the method of Durnin and Womersley [5], and an index of body build was calculated from weight and height ($kg \cdot m^{-2}$). A blood sample was drawn for determining hematocrit. The following tests of pulmonary function were performed using a Collins Spirometer Model 06031: forced vital capacity (FVC), forced expiratory volume at 1 s (FEV_1), peak expiratory flow rate (PEFR), forced expiratory flow (FEF_{25-75}), and maximal voluntary ventilation (MVV).

Subjects were instrumented for acquisition of an electrocardiogram (ECG), impedance cardiogram (ZCG), phonocardiogram, and carotid pulse contour, which were recorded on a Brush eight-channel recorder, and for manual monitoring of arterial pressure by sphygmomanometry. The test protocol consisted of subjects supine for 10 min, seated for 10 min, and standing for 10 min, in that order. Data were recorded during the final minute of each posture, and ten sequential cardiac cycles were analyzed and averaged from each recording by use of a dipitizing system similar to that described by Frey [6, 7]. Arterial pressure was measured once at each posture by a trained and experienced registered nurse.

Variables

We measured or calculated the following cardiovascular variables: heart rate (HR) from the ECG; thoracic impedance (Z_0), first derivative of change in impedance cardiogram (dZ/dt), ejection time (T), and stroke volume (SV) from the ZCG [1, 2]; systolic time intervals from the ECG, phonocardiogram, and carotid pulse contour including cardiac cycle interval measured between sequential ECG Q waves (Q-to-Q) and electromechanical systole (EMS) and left ventricular ejection time (LVET), which were used in the calculation of pre-ejection period (PEP) [6–9]; and systolic (SBP) and diastolic (DBP) arterial pressures.

Stroke volume was calculated as:

$$SV = \rho \cdot \frac{L^2}{Z_0^2} \cdot T \cdot \frac{dZ}{dt_{min}},$$

where ρ (ohm·cm) is resistivity of blood based on hematocrit [10]; L (cm) is length between inner electrodes measured on anterior midline body surface; Z_0 (ohm) is baseline impedance of the thorax; T (s) is ejection time measured from ZCG as horizontal distance from start of steep upstroke to downward deflection approximately concurrent with second heart sound (fig. 1), and $(dZ/dt)min$ ($ohm \cdot s^{-1}$) is the minimum value for dZ/dt during systole, measured as vertical distance from point where upward trace crosses baseline to peak (fig. 1).

Changes in SV from supine to sitting and supine to standing were calculated to determine whether relative changes in SV of sub-

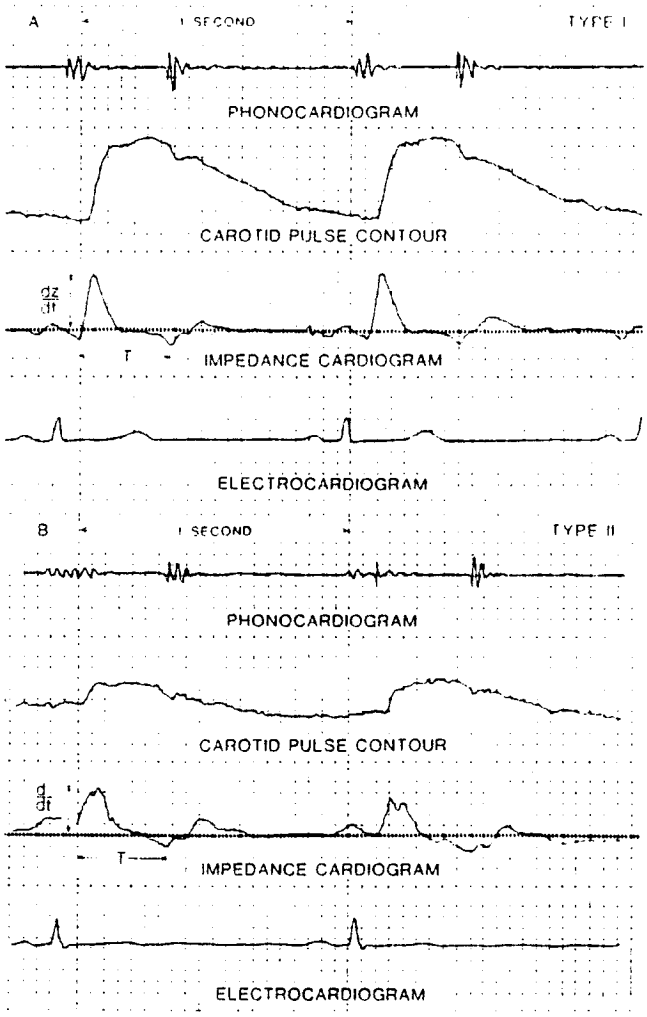


Fig. 1. Sample recording of phonocardiogram, carotid pulse contour, impedance cardiogram, and electrocardiogram. dZ/dt is measured from baseline to peak of impedance cardiogram, and ejection time (T) is measured from point of change in slope to nadir of wave at end of ejection. Figure 1A shows type I impedance cardiogram, and figure 1B shows type II.

jects exhibiting aberrant waveforms were similar to those of subjects with normal waveforms.

Cardiac output (CO = HR·SV) and mean arterial pressure [MAP = (1/3)SBP + (2/3)DBP] were calculated from the mean values for an individual at each recording period.

Recordings were visually examined, and data were inspected to reveal whether the bifurcated waveform could result from factors known to affect thoracic impedance. To determine if the double peak in the ZCG was related to a functional dissociation between the two ventricles, we measured the magnitude of the A2-P2 split in the second heart sound and compared it between beats with and those without a double peak in the ZCG. Beats at which bifurcated waveforms occurred were checked to determine if they occurred preferentially around 70 beats·min^{-1}, where orientation of erythrocytes has a maximal influence on the ZCG [3].

Data Analysis

Impedance cardiograms were classified as having either normal single-peak (type I) or bifurcated double-peak (type II) characteristics. Five individuals who were familiar with the impedance waveform graded the recordings. Subjects were classified as type II if they had type II waveforms in any posture. Sample traces of the two types of waveforms are presented in figure 1.

Data from men and women were analyzed separately. The numbers of subjects with type II waveforms in each posture were compared by χ^2 analysis. Physical, anthropometric, pulmonary, and cardiovascular characteristics of subjects exhibiting type I waveforms were compared with these characteristics of subjects exhibiting type II waveforms by T tests – as were changes in SV from supine to sitting and from supine to standing. Differences in cardiovascular variables as a function of impedance waveform type and posture were determined by analysis of variance. Alpha was set to 0.05.

Results

Eight men and 5 women had type II impedance waveforms. Four of these men had aberrant waveforms in two or three postures. The 5 women had aberrant waveforms in only one posture each. Aberrant waveforms occurred most often when subjects were standing, but this was not a statistically significant difference.

The physical, anthropometric, and respiratory characteristics of the men with type I waveforms and those with type II waveforms are compared in table 1. The 'type II' men were older and shorter and had more ponderous body builds, more body fat, and larger chests and abdominal circumferences than the 'type I' group. Their FVC and FEV_1, as well as their hematocrit, were lower than those of the 'type I' group. The women's characteristics are listed in table 2. The 'type II' women also had more ponderous builds, more body fat, and larger chest and abdominal circumferences; and they had a lower peak expiratory flow rate than the 'type I' women.

Values for cardiovascular variables of subjects with type I and type II waveforms and significance levels (p values) from the analyses of variance are listed in table 3 for men and in table 4 for women. Posture exerted a significant effect on all measured variables except the SBP of both men and women and magnitude of the (dZ/dt) trace of men. 'Type II' men had smaller SV, CO, (dZ/dt), and PEP but greater T than 'type I' men. 'Type II' women had faster HR (shorter Q-to-Q interval) than the 'type I' women.

An interaction between type and posture indicates cardiovascular responses to postural change of subjects with type II waveforms differed from those of subjects

Table 1. Physical characteristics of male subjects producing type I and type II impedance waveforms

Variable	Type I (n = 22)		Type II (n = 8)		I vs. II p
	mean	SE	mean	SE	
Age, years	37	2.3	47	2.7	0.02
Height, cm	179	1.0	175	1.5	0.03
Weight, kg	79	2.1	84	4.8	0.24
Body build index, $kg \cdot m^{-2}$	24.4	0.5	27.4	1.4	0.02
Body fat, %	23	1.3	31	2.1	0.01
Chest circumference, cm	98	1.7	105	3.7	0.05
Abdominal circumference, cm	88	1.7	98	4.9	0.02
Forced vital capacity, l	5.7	0.21	4.6	0.20	0.01
Forced expiratory volume at 1 s, l	4.4	0.16	3.6	0.16	0.01
Peak expiratory flow rate, $l \cdot s^{-1}$	10.9	0.41	10.6	0.70	0.65
Forced expiratory flow (25–75), $l \cdot s^{-1}$	4.1	0.25	3.5	0.33	0.18
Maximum voluntary ventilation, $l \cdot min^{-1}$	155	8.6	127	10.1	0.08
Hematocrit, %	46	0.5	43	0.6	0.02
Length between electrodes, cm	26.4	0.35	25.7	0.45	0.24

Table 2. Physical characteristics of female subjects producing type I and type II impedance waveforms

Variable	Type I (n = 25)		Type II (n = 5)		I vs. II p
	mean	SE	mean	SE	
Age, years	38	2.3	43	4.0	0.31
Height, cm	164	1.1	160	3.2	0.24
Weight, kg	61	2.4	68	2.2	0.22
Body build index, $kg \cdot m^{-2}$	22.7	0.8	26.4	1.1	0.05
Body fat, %	31	1.2	38	2.7	0.04
Chest circumference, cm	77	1.4	86	2.0	0.01
Abdominal circumference, cm	82	1.6	97	3.1	0.01
Forced vital capacity, l	3.7	0.1	3.4	0.3	0.28
Forced expiratory volume at 1 s, l	3.0	0.1	2.7	0.2	0.17
Peak expiratory flow rate, $l \cdot s^{-1}$	7.3	0.25	6.1	0.24	0.05
Forced expiratory flow (25–75), $l \cdot s^{-1}$	3.0	0.18	2.6	0.20	0.38
Maximum voluntary ventilation, $l \cdot min^{-1}$	116	4.3	111	7.4	0.62
Hematocrit, %	40	0.4	39	1.3	0.78
Length between electrodes, cm	25.8	0.4	25.6	0.8	0.86

with type I waveforms. Interactions between type and posture were observed for Q-to-Q interval, SV, (dZ/dt), EMS, and T in men and for Q-to-Q interval and thoracic impedance in the women.

Figure 2 illustrates the differences in pattern of responses to postural change between 'type I' and 'type II' subjects in Q-to-Q interval, a variable which is independent of the ZCG. Figure 3 illustrates the difference in pattern of responses to postural change between 'type I' and 'type II' men in SV, a variable which was determined from the ZCG. The increase in SV from standing to supine was significantly smaller in 'type II' (28 ml) than 'type I' men (45 ml).

Type II waveforms (18 recordings) occurred at HRs ranging from 54 to 100 beats·min^{-1}, without preference. Almost all bifurcations occurred at, or occasionally after, the end of the first heart sound, in the ejection phase of the cardiac cycle and not during the pre-ejection period. Duration of the A2-P2 split of the second heart sound did not differ between normal and aberrant beats; however, the first heart sound appeared prolonged in some type II recordings (fig. 1).

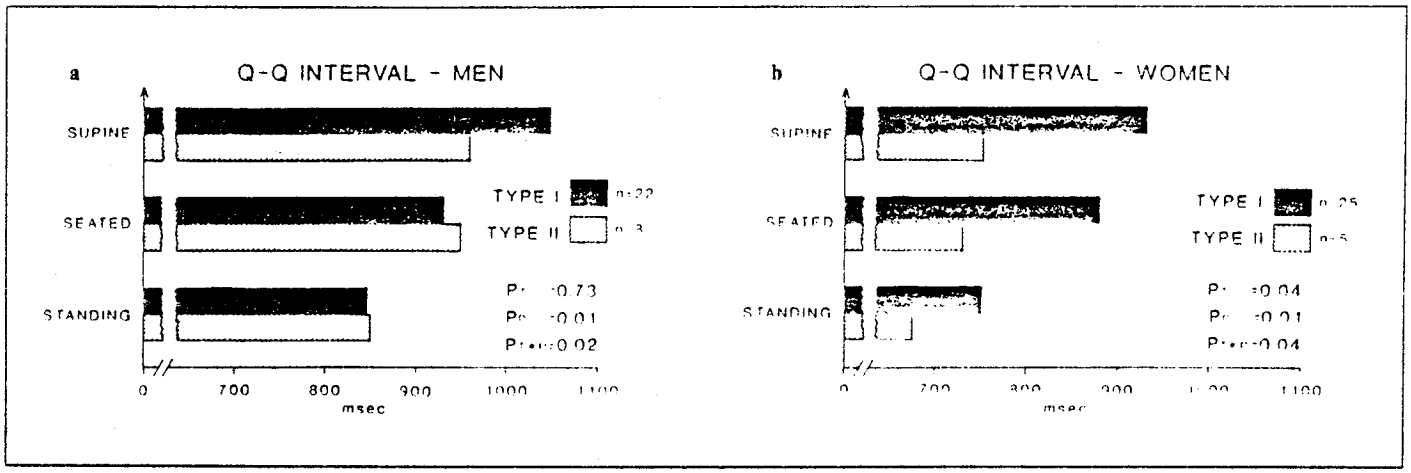
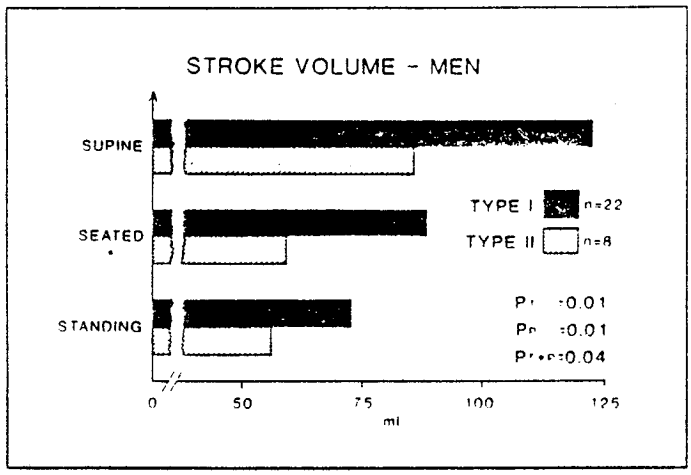


Fig. 2. Q-Q interval in supine, seated, and standing postures for subjects producing type I (dark bars) and type II (hatched bars) impedance cardiograms. Pt is significance level (p) for type, Pp is p for posture, and Pt·p is p for interaction of type and posture. Figure 2a shows data for men and figure 2b for women.

Table 3. Cardiovascular variables for male subjects (mean ± SE)

Variable	Supine	Seated	Standing	Significant differences ($p < 0.05$)		
				type	posture	type-posture interaction
Type I (n = 22)						
Heart rate, beats·min^{-1}	59 ± 1.6	66 ± 1.8	72 ± 1.9		*	
Q-to-Q interval, ms	1,041 ± 30.3	927 ± 26.9	844 ± 23.9		*	*
Stroke volume, ml[a]	118 ± 6.3	87 ± 8.4	72 ± 3.8	*	*	*
Cardiac output, l·min^{-1}[a]	6.8 ± 0.4	5.7 ± 0.3	5.2 ± 0.3	*	*	
Systolic pressure, mm Hg	120 ± 2.0	120 ± 2.1	119 ± 2.3			
Diastolic pressure, mm Hg	77 ± 2.2	82 ± 1.9	84 ± 1.7		*	
Mean arterial pressure, mm Hg	91 ± 2.0	95 ± 1.9	96 ± 1.8		*	
Thoracic impedance, ohm	22.2 ± 0.6	24.9 ± 0.6	25.6 ± 0.6		*	
dZ/dt, ohm·s^{-1}[a]	1.83 ± 0.10	1.88 ± 0.09	1.87 ± 0.08	*		*
Electromechanical systole, ms[b]	398 ± 5.0	376 ± 5.8	350 ± 5.1		*	*
Left ventricular ejection time, ms[a]	310 ± 4.4	278 ± 5.5	247 ± 4.7	*	*	*
Pre-ejection period, ms[b]	94 ± 2.6	109 ± 3.0	112 ± 3.2	*	*	
Type II (n = 8)						
Heart rate, beats·min^{-1}	63 ± 4.0	65 ± 4.7	74 ± 5.8		*	
Q-to-Q interval, ms	967 ± 49.7	945 ± 57.9	845 ± 64.7		*	*
Stroke volume, ml[a]	84 ± 9.6	59 ± 6.9	56 ± 5.9	*	*	*
Cardiac output, l·min^{-1}[a]	5.2 ± 0.6	3.8 ± 0.4	4.0 ± 0.4	*	*	
Systolic pressure, mm Hg	127 ± 3.0	125 ± 4.0	125 ± 4.6			
Diastolic pressure, mm Hg	80 ± 3.1	83 ± 3.1	86 ± 3.0		*	
Mean arterial pressure, mm Hg	96 ± 2.9	97 ± 3.2	99 ± 3.4		*	
Thoracic impedance, ohm	22.6 ± 0.4	24.7 ± 0.4	25.8 ± 0.4		*	
dZ/dt, ohm·s^{-1}[a]	1.50 ± 0.15	1.27 ± 0.16	1.45 ± 0.09	*		*
Electromechanical systole, ms[b]	379 ± 9.7	368 ± 10.8	347 ± 11.8		*	*
Left ventricular ejection time, ms[a]	311 ± 13.6	315 ± 13.0	280 ± 16.2	*	*	*
Pre-ejection period, ms[b]	80 ± 3.7	95 ± 3.2	104 ± 5.4	*	*	

[a] Calculated from impedance cardiogram.
[b] Calculated from electrocardiogram, phonocardiogram, and carotid pulse contour.



Discussion

We have identified apparently healthy individuals who produce aberrant waveforms when examined with impedance cardiography. We have observed differences between the group of individuals who produced aberrant waveforms and the group of individuals who produced normal waveforms in values for the cardiovascular variables that are derived from this waveform. And we have identified differences between the groups in physical, anthropometric, pulmonary, and cardiovascular charac-

Fig. 3. Stroke volume in supine, seated, and standing postures for men producing type I (dark bars) and type II (hatched bars) impedance waveforms. Pt is significance level (p) for type, Pp is p for posture, and Pt·p is p for interaction of type and posture. (Stroke volumes did not differ between women with type I impedance cardiograms and those with type II.)

Table 4. Cardiovascular variables for female subjects (mean ± SE)

Variable	Supine	Seated	Standing	Significant differences ($p < 0.05$)		
				type	posture	type-posture interaction
Type I (n = 25)						
Heart rate, beats·min^{-1}	67 ± 2.4	70 ± 2.2	82 ± 2.6	*	*	
Q-to-Q interval, ms	930 ± 32.7	879 ± 28.7	752 ± 23.3	*	*	*
Stroke volume, ml[a]	82 ± 3.5	62 ± 3.2	48 ± 2.6		*	
Cardiac output, l·min^{-1}[a]	5.4 ± 0.3	4.3 ± 0.3	4.0 ± 0.3		*	
Systolic pressure, mm Hg	108 ± 2.5	111 ± 2.4	110 ± 2.2			
Diastolic pressure, mm Hg	70 ± 1.5	76 ± 1.6	78 ± 1.8		*	
Mean arterial pressure, mm Hg	83 ± 1.6	88 ± 1.6	88 ± 1.8		*	
Thoracic impedance, ohm	30.4 ± 0.8	33.0 ± 1.0	34.3 ± 1.0		*	*
dZ/dt, ohm·s^{-1}[a]	2.82 ± 0.13	2.68 ± 0.14	2.66 ± 0.13		*	
Electromechanical systole, ms[b]	400 ± 6.2	388 ± 5.6	352 ± 5.7		*	
Left ventricular ejection time, ms[a]	318 ± 5.4	294 ± 5.5	251 ± 5.5		*	
Pre-ejection period, ms[b]	96 ± 2.7	105 ± 3.0	112 ± 3.0		*	
Type II (n = 5)						
Heart rate, beats·min^{-1}	80 ± 1.7	82 ± 2.8	89 ± 2.2	*	*	
Q-to-Q interval, ms	754 ± 16.8	734 ± 26.4	675 ± 16.6	*	*	*
Stroke volume, ml[a]	68 ± 6.4	48 ± 2.1	42 ± 4.1		*	
Cardiac output, l·min^{-1}[a]	5.5 ± 0.5	4.0 ± 0.2	3.8 ± 0.4		*	
Systolic pressure, mm Hg	118 ± 3.6	117 ± 2.1	118 ± 4.3			
Diastolic pressure, mm Hg	74 ± 4.9	80 ± 4.3	80 ± 4.2		*	
Mean arterial pressure, mm Hg	89 ± 4.4	93 ± 3.1	93 ± 4.1		*	
Thoracic impedance, ohm	31.1 ± 1.0	31.8 ± 0.9	33.7 ± 1.1		*	*
dZ/dt, ohm·s^{-1}[a]	2.58 ± 0.29	2.06 ± 0.19	2.44 ± 0.24		*	
Electromechanical systole, ms[b]	381 ± 6.6	364 ± 6.1	335 ± 3.7		*	
Left ventricular ejection time, ms[a]	311 ± 10.8	294 ± 18.6	240 ± 3.8		*	
Pre-ejection period, ms[b]	92 ± 4.5	101 ± 5.4	103 ± 4.6		*	

[a] Calculated from impedance cardiogram.
[b] Calculated from electrocardiogram, phonocardiogram, and carotid pulse contour.

teristics, and cardiovascular responses to postural change, that were determined independently of this waveform.

The aberration that we have observed is in the dZ/dt peak of the ZCG, which Kubicek et al. [1, 2] have shown occurs simultaneously with maximal blood flow velocity in the ascending aorta as measured by electromagnetic flow probe. We have considered both physics and pathology in an attempt to explain the occurrence of this bifurcated waveform in these subjects.

Changes in blood flow velocity affect orientation of erythrocytes. In a test tube, changes in flow velocity can cause a notched impedance trace [3]. In the body, flow velocity is a function of heart rate, and according to Lamberts et al. [3], the effect on impedance should be maximal at about 70 beats·min^{-1}. However, our data showed no preferential rate for bifurcated beats throughout the range of 50–100 beats·min^{-1}.

Left ventricular outflow is the major determinant of the dZ/dt peak in the impedance waveform; but blood flowing in the atria, venae cavae, and pulmonary circulation also affects the ZCG. If blood flow in these regions exerted an effect at mid-ejection in a direction opposite that of left ventricular ejection, it could produce the aberrancy. Or, if ejection itself caused another event in the thorax at mid-ejection, such as shifting the heart to facilitate venous return, this might affect the waveform. Or, if orthostasis introduced an impediment to outflow, the bifurcation might occur. While we cannot rule out these possibilities, we have no explanation of why they would occur.

Our subjects who produced type II waveforms appeared to be healthy. Their medical histories were examined by a physician who found no evidence of cardiovascular or other disease that would explain their impedance waveforms. No obvious evidence of cardiovascular pathology exists in the ECGs or PCGs recorded during this study. We found no collaborative evidence that the ventricles were beating asynchronously. Furthermore, S-T segment depression was not observed in the ECGs (standing at rest) of 2 subjects, each of whom had had five normal clinical stress tests previously.

However, a bifurcated waveform similar to that we have observed has been previously reported for patients with cardiovascular pathology, including severe mitral insufficiency [1] and mitral stenosis complicated by tricuspid incompetence [11]. Luisada et al. [12] observed bifurcated ZCGs from some patients with right or left bundle branch block, but they concluded that the double peak resulted not from a nonsimultaneous output due to the block but from a dyssynergy in left ventricular contraction due to scarring left from a myocardial infarction. They do not explain the bifurcations in impedance cardiograms from 2 of their normal subjects, both over 80 years of age. (Our 'type II' subjects ranged from 32 to 55 years.) Bifurcated waveforms have also been recorded from patients with heart failure [13] and from patients who have undergone 3 h of hemodialysis [4]. Patients with complete A-V block, however, did not produce bifurcated waveforms [14, 15]. Previously, clinical investigators have found greater diagnostic value in the diastolic portion of the ZCG [13, 16]; however, the dZ/dt peak may become diagnostically important.

The bifurcated ZCG is not a transient characteristic. We had recorded ZCGs from 2 of our male subjects several years before, and they produced the same type waveform then – at several examinations. Furthermore, the aberrancy was not evident at supine rest but occurred during lower-body negative pressure, which is an orthostatic type of stress similar to standing.

Since SV is derived from parameters that are altered in the aberrant waveform, we would expect a concomitant change in computed SV, and the 8 men who produced type II waveforms in this study had a lower mean SV than those with type I waveforms. However, they also had a different pattern of change in SV between postures. We are not certain whether they actually responded differently or we failed to determine their SV accurately.

Despite our inability to explain the aberrant waveforms on the bases of physics or pathology, the individuals who produced these waveforms (especially the men) differed from other subjects. They were as a group older. They were shorter and stockier and had more body fat. Their pulmonary function, possibly reflecting their age and height, was lower. The 2 men who had been previously tested on a treadmill had very low V_{O_2} peak (less than 30 ml·kg^{-1}·min^{-1}), which may indicate people who have a low level of aerobic fitness are more prone to this type of aberrant waveform.

Several of the cardiovascular differences that were shown between 'type I' and 'type II' groups are independent of the ZCG. 'Type II' women had faster heart rates than 'type I', and 'type II' men had shorter pre-ejection periods. Either of these characteristics could indicate a higher baseline level of sympathetic nervous system outflow in subjects producing type II waveforms. When they changed posture, both male and female 'type IIs' experienced less change in Q-to-Q interval than their 'type I' counterparts. The women producing type II waveforms

also had less change in thoracic impedance with posture change, which may indicate they experienced less reduction in thoracic fluid upon standing and thereby explain their lesser heart rate response.

Thus, real differences existed between the subjects producing type II waveforms and the other subjects. Although we recruited 'normal, healthy' subjects, we acknowledge that this bifurcated impedance waveform may indicate something is other than 'normal'. Therefore, we recommend that neither absolute values nor relative changes in SV from bifurcated impedance cardiogram waveforms be extrapolated to the general population until we understand the etiology of these waveforms.

Most individuals produce normal ZCG waveforms, and impedance cardiography continues to be a valuable technique for use in the applied physiology laboratory.

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